Noyes (H. D.)

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BY

HENRY D. NOYES, M.D.

NEW YORK

Reprinted from the MEDICAL RECORD, *April* 4, 1891

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HEMIANOPSIA.[1]

THE subject which I am to consider is a visual manifestation of intra-cranial disorder. My remarks will relate to the visual disturbance rather than to the cerebral pathology. I ask more critical attention to this symptom in the hope that light may be shed upon the intricate problems of cerebral disease. The aim is to reach greater precision in cerebral localization, by closer study of visual symptoms.

Let us briefly sketch the course of the visual tract from the retina to its ultimate destination in the brain. The peripheral percipient elements in the retina are the cones and rods, which are connected by fibres with the outer and inner granule layers and by successive fibres with the ganglion cells; from these proceed the optic nerve fibres. The number of cones and rods is estimated to be seven times as numerous as the optic nerve fibres, and the estimate of optic fibres is between four hundred and thirty-eight thousand (Salzer) and one million (Krause).[2] The optic nerve fibres lie in layers, but in the region of the macula lutea they are finer and in fewer layers, and anastomose freely. At this space the bundles into which they are collected, cannot, as elsewhere, be separately traced. The macular fibres are the most important in function, and are clustered into a triangular bundle composing about one-fourth of the nerve, which

[1] Read before the New York Academy of Medicine, February 8, 1891, and illustrated by lantern slides.

[2] For these statements see Wilbrand die hemianopische Gesichtsfeld Formen, Wiesbaden, 1890.

enters the papilla at its infero-temporal side. As it pursues its way through the orbital portion, it gradually approaches the axis of the nerve, which it reaches at the optic canal. At the front of the chiasm it occupies its upper and inner portion, but in the tractus it sinks to the central portion and remains there until it arrives at the brain. The semi-decussation of the optic tract in the chiasm is an accepted fact, despite the incredulity of Michel. The papillo-macular bundle is likewise divided into crossing and direct fibres which at the anterior part of the chiasm can be separately distinguished, but at its posterior part and in the tractus become inextricably mingled. The macular fibres anastomose with each other in the retina and they have additional opportunity to do this in the chiasm and the tractus. The tractus winds around the crus cerebri and terminates in two roots upon the corpora geniculata externa and interna and upon the posterior part of the optic thalamus, called the pulvinar. Fibres also go to the anterior pair of the corpora quadrigemina, but these organs are not regarded as concerned in vision, but in the activity of the pupil. The parts just referred to are called the primary visual ganglia.

In them we find innumerable ganglion cells in which fibres of the tractus lose themselves, and thereafter a new set of fibres proceeds backward through the posterior part of the internal capsule to the cortex under the name of the visual radiation, or fibres of Gratiolet or of Wernicke. Passing through the internal capsule they cross the sensitive fibres coming down from the hemisphere, are rather closely massed, and then, spreading out like a fan, rise upward, wind outside the tip of the lateral ventricle to reach their destination at the lower part of the median surface of the occipital lobe.

Varieties of Hemianopsia.—In all cases the dividing line is either horizontal or vertical, the definition not being very exactly interpreted. There may be loss of half the field in only one eye and this points out a lesion of one optic nerve in front of the chiasm. If both upper

or both lower half fields are wanting, the seat of lesion will be on the lower or upper part of the chiasm, or of both optic nerves. A case very briefly reported in the *British Medical Journal* for November 22, 1890, had loss of the upper half of the field in one eye, and of the lower half in the other eye. This requires a double lesion in front of the chiasm. Total blindness of one eye combined with loss of the half or of the quarter of the field in the other is explained by a lesion of both nerves, on one side complete, on the other side incomplete. All these are peripheral lesions; and the same is true of the cases of bi-temporal and bi-nasal hemianopsia. Bi-temporal half blindness is explained by a lesion at the middle of the chiasm involving the crossing fibres from both tractus. It may be from tumor (Weir Mitchell), it may be from fracture at the base of the skull, etc.

The excessively rare condition of bi-nasal hemianopsia presupposes a lesion on both sides of the chiasm, or on the outer side of each optic nerve and impairs the direct fibres (Knapp).

We are chiefly interested in another variety of hemianopsia, viz., that which affects the same side of each field and in which the dividing line is nearly vertical. This variety, called homonymous, implies injury of the visual tract behind the chiasm. On this subject most admirable papers have been written, by Starr, 1884; by Seguin, 1886 to 1887; by Hun, 1887; and by Swanzy,[1] in which are set forth other distinctive and localizing symptoms which accompany the visual defects by which the focus of the disease may be more or less accurately determined. To these symptoms I shall only incidentally refer. My purpose is to call attention to some peculiarities in the fields of the vision which have not been generally attended to. I am indebted for much of what I offer to the last monograph by Wilbrand, an atlas of hemianopic defects, 1890, Wiesbaden.

[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. ix., 1889.

The dividing line in hemianopsia is sometimes precisely on the vertical meridian and straight. That it is many times not on the vertical meridian and not straight has long been observed.[1] This occurs both in the peripheral, *e.g.*, bi-temporal and in the central forms. It may bend around the point of fixation upon the blind side, and thus greatly mitigate the injury inflicted on sight by sparing the whole of the macula lutea. That the boundary of the blind half may not for a considerable extent reach the vertical meridian and may be a sinuous or oblique line, or form an open angle, has also been noted. Wilbrand points out that in 77 cases of homonymous hemianopsia the dividing line was central in 29 cases. In 33 the blind portion was smaller than the seeing portion of the retinæ and to an equal extent in both eyes. In 5 cases the dividing line was in one eye central and in the other eye was pushed away from the fixing point toward the blind side. In 10 cases the amount of displacement of the dividing line beyond the vertical meridian was unlike in the two eyes. The blind side is always either smaller in area than the seeing side, or may be equal to it. It is never greater.

Out of 32 cases of bi-temporal hemianopsia in only 9 did the boundary coincide with the vertical meridian. In 12 total blindness of one eye precluded comparison. In 20 cases the blind sides were the smaller. We cannot, at present, draw any practical deductions from these facts, and in explanation of them we have to refer to the anastomosis of the fibres from each tractus in the retinæ as well as in the optic nerves and chiasm where crossing and direct fibres lie in close relation to each other.

But a new significance attaches to this circumstance since the publication within the year of two cases of double hemianopsia, one by Forster,[2] and another by Schweigger.[3]

[1] See Schweigger: Graefe's Archiv, vol. xxii., abth. iii., p. 276, 1876.
[2] Graefe's Arch. für Oph., vol. xxxvi., abth. 1, 1890.
[3] Archives of Ophth., 1891.

Forster's case was a man who at forty-four years of age had from apoplexy complete homonymous hemianopsia on the right side, with preservation of 1° or 2° of the field toward the blind side, at the macula.

He had another attack five years later, which befell the opposite side and caused complete loss of the remaining halves of each visual field. The second attack did not reach completeness until after three days. When Professor Forster examined the man six weeks later he seemed to be totally blind in every sense. His pupils were about four millimetres wide and gave a feeble reaction to light.

This led to inquiry as to possible vision, and it was found that he could read Snellen $1\frac{1}{2}$ very slowly with each eye; distant vision was about $\frac{1}{10}$. Perimetric examination revealed the preservation in each eye of a central field measuring about $2\frac{1}{2}°$ vertically by 3° horizontally and below the horizontal meridian. It would cover a surface 13.5 ctm. (about $5\frac{1}{4}$ inches) in diameter at the distance of 3 m. There was no perception of color. The ophthalmoscopic appearances were normal.

Schweigger's case was a man seventy-five years of age, whom he had treated for years, for chronic conjunctivitis and incipient cataract. I have been permitted to quote it from advance proof sheets by the kindness of Dr. Knapp. In September, 1888, he had an apoplectic attack with left homonymous hemianopsia, not accompanied by other symptoms. In August, 1889, there came another attack with complete homonymous hemianopsia of the opposite side. The whole of both visual fields was destroyed, except a central space in each eye measuring 2° or 3° in diameter. He had retained as the issue of his first attack vision of $\frac{9}{12}$ and $\frac{6}{36}$ in the respective eyes, and this was not made worse by the second attack. Within these fields there was correct color perception. Over the right half fields, the seat of the last attack, there was some perception of light. By the ophthalmoscope nothing abnormal was to be seen.

Still another case is given by Moeli,[1] which occurred in a man affected by dementia paralytica. He had double hemianopsia at successive periods, but on account of his defective mental state an accurate examination of his fields could not be made. But it was evident that he could see within a very small field.

A case by Bouveret [2] of double lesion of the occipital lobes was so soon followed by death as to preclude careful examination of vision. No other functions were damaged. It is not easy to explain the remarkable facts of these cases. Evidently no explanation can be based upon the anastomosis of optic nerve fibres in any of the peripheral parts of the visual apparatus. We have to do with the preservation of that region of the cortical visual centre which supplies the macula lutea. It has been an open question whether the cortical visual region corresponds topographically to the retina and field of vision, and only within four years has any positive evidence been gained. The case published by Hun showed that lesion of the lower half of the cuneus causes loss of function of the upper quadrant of the retina of the same side, *i.e.*, the lower quadrant of the corresponding field is darkened. The demonstrative character of this case is unquestionable, in regard to a topographical correspondence between the retina and the cortex. Whether the inference can be carried to minute details will be considered presently. The cases of Forster and Schweigger are significant, but not demonstrative. Even should an autopsy be obtained in either it would probably prove impossible to identify the intact regions of the cortex. How they can remain intact when the remainder of the region has lost its nutritive supply, is, of course, conjectural. Forster supposes that there must be a special arrangement of vessels to nourish the macular region of the cortex independent of the posterior cerebral arteries and thinks it may come

[1] Archiv f. Psychiat, xxii., 1, 101.
[2] Revue Générale d' Ophthalmologie, p. 481, 1887.

from fine vessels in the pia mater. An attentive anatomical study of this point might give us information.

There are other cases on record of double hemianopsia, but with no note that any vision remained. More light as to the topographical relation between the retina and the cuneus may come from a counterpart to the cases of Forster and Schweigger.[1] A healthy man, aged forty-five, suddenly found reading confused and difficult without any impairment of distant vision. He could read Snellen 1, although not easily and preferred the small words. Color sense was unimpaired. By the ophthalmoscope nothing abnormal. Their were no cerebral symptoms. Pupils and eye muscles normal; urine negative. By the perimeter there was found a small area of blindness on the right half of each field just touching the macula, about equal to twice the diameter of the optic disk, and in outline and extent precisely alike. They were delineated by a test object 1 mm. square. These isolated twin defects remained unchanged for four years under repeated examinations. Their presence was only embarrassing in near work, and in ordinary vision they were unnoticed. In this case there can hardly be any other explanation of the lesion than that it was situated in the cortex, because it was so minute and so well defined and was sudden in its onset. In the medullary (sub-cortical) substance it would hardly have been so small. Its presence seems to compel the inference that the retina and the cortex answer to each other with a remarkable degree of detail. Further proof is to be found in another case (Fig. 34, Taf. x., Wilbrand). A man aged thirty-four had headache for years, the pain not very severe and not to be precisely located. No vomiting or nausea. Six weeks before being examined he awoke with serious disturbance of sight and noticed with closed eyes a brilliant flashing on the right side of each field. V. $= \frac{11}{20}$; the fundus both normal; urine specific gravity 1030, but without sugar. By the

[1] Reported by Wilbrand, loc. cit., p. 5.

perimeter was found an insular defect of oval form in each right field on the horizontal meridian, at the same distance in each eye from the fixation point. The patient was seen only once.

Another case by Wilbrand is figured at three successive periods within five years, during which time only slight changes occurred in the homonymous defects. They reached from the fixation point to the twenty-third circle of latitude on the left side. The only symptom was severe headache. After five years sugar appeared in the urine.

In all these cases of small homonymous, permanent, and similar visual defects, unaccompanied by serious cerebral symptoms, the evidence points almost certainly to a small lesion of the medullary or of the gray matter in the visual centre.

The quadrant defects, such as Hun has reported, are not very rare. I have seen two cases. In one it was not exactly bounded, because a perimeter could not be used in the patient's bed-room. He was a large healthy man, aged sixty-three, a distinguished lawyer. He had for eight years had hyaline casts in the urine and was accustomed to examine for them with the microscope. Fourteen years previous he had a small hemorrhage at the macula lutea of the right eye which caused metamorphopsia, but the injury to sight was not permanent—and while a slight zigzag distortion could be noted in fine parallel lines, the defect was not annoying. In April, 1889, he had a cerebral attack whose symptoms were vague, and which came after he had for two days noticed vortices and muscæ floating before him. I was called to him ten days after and found him much depressed in mind ; able to converse intelligently, although speech was more than usually slow. He evidently had difficulty in collecting his thoughts. His chief complaint was of confusion of sight and inability to read. There was no paralysis of motion or sensation. There was no headache. There had been pain in the lumbar region. He recog-

nized objects perfectly, could call their names, and remembered the events of the preceding days. His manner was very different from his usual intelligent, although deliberate, character. There was no diplopia; to the ophthalmoscope the eyes were normal. Testing the visual fields with the hand there was found homonymous hemianopsia of the upper two-thirds of the left side. The defect did not include all of the left lower quadrant, and the boundary line could be moderately well defined. I did not see him afterward. He died about seven months later from evident brain lesion, stated to be with signs of softening. There was no autopsy.

Another case of quadrant homonymous hemianopsia which I have noted is as follows: The defect really includes the upper two-thirds of the right half fields, taking in 110°. The man, aged forty, printer, seen at the New York Eye and Ear Infirmary in May, 1887. Indulges freely in alcoholic drink, had been a soldier, and received various slight injuries. Has scars on the head, one over right parietal eminence, one on each side of frontal bone, near fronto-parietal sutures. These were probably received during some of his sprees. Within two years had much headache; denies syphilis and there are no symptoms. On Sunday, May 8, 1887, after having been drunk, noticed that he did not see objects on his right side. Complained that he was afraid of being run over in the street. On examination found to be very myopic, O. D. — 15 D., V. = $\frac{20}{100}$. In the fundus extensive choroidal lesions, both circumpapillary and diffused. In the fields is a homonymous partial sector like hemianopsia on the right side. In the left eye the defect reaches over into the adjacent left side, but here the visual perception remains to a certain degree and is fully accounted for by choroidal changes seen by the ophthalmoscope in the corresponding part of the fundus. Patient is also deaf. He has been observed up to September, 1890, and the visual defect remains unchanged. He suffers constantly from vertigo, which might, perhaps, be attributed to his

aural disease, were it not that the symptom has appeared since the eye trouble occurred.

In a case reported by Brill[1] the symptoms were partial hemiplegia and hemianæsthesia on right side, amnesic aphasia and total loss of color perception in both eyes. There must have been right hemiopia although it was not investigated. The lesion was a softened area at apex and lower part of left cuneus, and in adjacent part of gyrus lingualis. It reached within 1.5 ctm. of tip of occipital lobe. The double achromatopsia has great interest and is to be considered in connection with Verrey's case (*vide infra*).

These cases, because of the absence of other cerebral symptoms, point to a lesion of the mesial portion of the occipital lobe. Sector defects may, however, arise from lesions in other parts of the brain. For example, Schweigger[2] gives the case of a woman who, after childbirth fifteen months previous, had hemiplegia and slight aphasia and alexia. All these symptoms passed away. There remained a loss of field on the right side of each eye on the horizontal meridian which included a sector about 20° in breadth. The location of the lesion could not have been cortical exclusively. It may have been subcortical.

A case of sector hemianopsia from tractus lesion is given by Marchand.[3] A student, aged twenty-one years, in blooming health, was examined for myopia and found to have slight double optic neuritis and V. = $\frac{1}{3}$. He was under observation fifteen months and improved in sight nearly to the normal. On his last visit a defect in the upper left quadrant of each eye was discovered. Ten days afterward severe headache occurred, some strabismus, vomiting, coma, and death. At the autopsy a glioma was found in the right temporal lobe near the gyrus hippocampi. It encroached on the outer surface of the tractus at its posterior part, which was softened and red-

[1] American Journal of Neurology, February, 1883.
[2] Archiv f. Ophthal., Bd. xxii., Abth. iii. p. 297.
[3] Graefe's Archiv, Bd. xxviii., Abth. 2, p. 64, 1882.

dish yellow, while the rest of it was normal. The defect in the field was caused by the lesion of the tractus.

Another and very recent case of quadrant homonymous defect due to lesion of one tractus is reported by Norris.[1]

In this case there were other symptoms besides those of visual disturbance, viz., muscular twitchings, impaired grip of left hand, partial loss of control of left leg, drowsiness, the Wernicke hemianopic pupillary inaction not obtainable, diplopia occurred, and finally complete paralysis of left side. The diagnosis was tumor of the base near the thalamus. At the autopsy a tumor was found at the junction of the right crus cerebri with the temporal lobe. By sections transversely the tumor was found to occupy the white matter within the right occipital and temporal lobes, reaching posteriorly a point just outside the descending horn of the lateral ventricle, anteriorly, and internally at the base it extended to the tuber cinereum and involved the optic tract. Above it reached close to the gray matter of the insula, but did not involve it. It rose about half an inch above the level of the pulvinar; it involved part of the lenticular nucleus, the anterior portion of the internal capsule, and extended laterally to the thalamus. It proved to be a glioma. The complete involvement of the right optic tract in the tumor accounted for the quadrant and otherwise irregular impairment of the visual fields. They were not symmetrical. In the left side more than a quadrant was destroyed and in the right side almost a half, whose border was both oblique and irregular. The extension of the tumor back into the white substance of the occipital lobe doubtless had much to do with the irregular hemiopia.

It therefore follows that absolute localizing value cannot be attributed to sector lesions of the fields. Yet the great probability favors the cortex. In this case the optic neuritis was the only sign until the close of the disease

[1] Trans. Am. Ophth. Soc., 1890, p. 470.

of the nature of the mischief, and this had, at that time, disappeared.

I might call attention to very irregular, but symmetrical types of visual defect. They are found in Schweigger's paper and are quoted in Wilbrand. I have some of my own experience. They may be omitted, except certain very peculiar kinds of which one is noted by Wilbrand and described at length.[1] A man, age not given, short and stout, who had had glycosuria, complained since October, 1888, of dizziness and frontal headache and confused sight. He could see parts of words, and in faces some of the features would be indistinct or invisible and other features clear. The symptoms increasing, he was confined to bed a few days, and during this time visions constantly presented themselves on *the left side*, both with closed and open eyes. He saw landscapes, heads, cats, furniture, etc. The illusions disappeared in a few days. When carefully examined on November 8, 1888, pupils, lids, eye muscles, and fundus were normal. There was correctible myopic astigmatism. The perimeter showed left-sided hemianopsia, and by using a small test object 5 mm. square, it was found that the blind half-fields were composed of innumerable insensitive areas separated by openings through which the object could be discovered. They could not all be mapped out, but one notable scotoma below the horizon is outlined ; the heavy, straight, meridian lines show places where the object was seen uninterruptedly. On the 26th of the same month, with increasing general symptoms, an apoplectic attack occurred with right hemiplegia, coma, and death.

Autopsy, twenty hours after. Atheroma of vessels at the base. In removing the brain the hemispheres were torn off at the peduncles, and while both were fragile, the right peduncle showed distinct yellow softening. The chiasm and tractus were normal. The same was true of the cortex, except at the tip of the right occipital lobe,

[1] Loc cit., p. 54, Taf. ix., Fig. 22.

which was softened. The softening occupied the third occipital convolution on the upper and medial surface, but below it reached forward to the fusiform convolution and the gyrus lingualis. Section disclosed a hemorrhagic softening, the size of a hazel nut, in the substance of the medullary matter, and the superjacent cortex had undergone yellowish discoloration. There were no lesions of the internal capsule or of the primary ganglia. The concentric limitation of both the right half-fields must be ascribed to the implication of the whole brain, and especially the other occipital lobe, in functional disturbance.

Wilbrand calls attention to the hallucinations which in his case appeared only in the affected half fields and quotes similar cases from Henschen and Putzel.[1] Dr. Petersen has in two papers recently recorded a similar occurrence. The case of Henschen presented lesions of the cuneus and lingual lobe, and that of Putzel involved the cuneus extensively, as well as the internal capsule and the outer border of the thalamus.

In subcortical lesions, as well as in cortical lesions, hemianopsia may be either complete or incomplete, but Wilbrand's case, so far as it goes, indicates that extreme irregularities in the fields are likely to be due to subcortical lesions.

Another feature of hemianopia requires consideration. The function of sight includes three subdivisions : Perception of light, of color, and of form. If the first be wanting, all are wanting. Given the first, either or both the remaining two may be lacking. Illustrations of such defects occur in hemianopsia. Hemianopic loss of color sense in both eyes is the most frequent among these rather rare cases. Of these, Swanzy [2] enumerates eight in literature and in one case there was an autopsy. Verrey reports it in *Archives d'Ophthalmologie*, tome viii., No. 4, 1888. A lady, aged sixty, had an apoplectic at-

[1] New York Medical Journal, August, 1890, and January, 1891.

[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. ix., 1889, p. 23.

tack, from which she fully recovered, except from the disturbance of sight. There was found absolute color blindness in the right half of each field, with reduced, but not complete loss of light and form sense in the same regions. There was no dyslexia nor mental blindness. During twenty months the condition was found to be the same. Finally she died of another apoplexy. I quote Mr. Swanzy's account of the autopsy: "The cause for death was found in a fresh hemorrhage in the right centrum ovale and lateral ventricle, while an old hemorrhagic cyst in the lower part of the left occipital lobe, extending into the temporal lobe on the mesial side, explained the hemiachromatopsia. This cyst was situated between the floor of the posterior horn of the left lateral ventricle and the basal surface of the occipital lobe. It had occupied the white substance of the inferior occipital convolution, and had almost completely destroyed the white substance of the posterior extremity of the occipito-temporal convolutions as well as that of the postero-inferior part of the cuneus. The cyst came nearly to the surface of the cuneus and of the occipito-temporal convolutions, having destroyed the deeper layers of their cortex."

Another case by Siemerling (see Fig. 45, quoted by Wilbrand, loc. cit., p. 144) presented on the right side complete hemianopsia combined with total loss of color sense on both left sides. There was mental blindness, and agraphia and amnesic aphasia. Vision was $\frac{1}{30}$. Within three and a half months vision improved to $\frac{3}{5}$. Color perception returned to the left sides and the mental blindness, alexia, and agraphia disappeared. The right hemianopia continued. There were no other symptoms.

A case of Schoeler's [1] presents on the left side of each field complete color blindness, with the addition in the left half of the right eye of a sector at its lower part where there is no light sense. Patient had bad headache, vertigo, attacks of half sight, swimming before the eyes.

[1] Bericht, 1884, p. 64.

No organic lesions. After three months the defect in lower part of the field of the right eye disappeared, but the loss of color sense in the left half fields persisted.

The loss of form sense with retention of light sense is to be determined by testing with letters or simple figures held eccentrically. This examination is not readily made, because we have not a standard in habitual use. One may test by holding up fingers. An abatement in form of sense can sometimes be ascertained.[1]

A case from Schoeler and Uhthoff[2] is in point. A man, aged sixty-eight, double hemianopsia. O. D., fingers at ten feet; O. S., fingers at twelve feet. Fundus normal. In right eye central opacity of cornea. Seven days previous was attacked by severe pain and congestion in the head, until his face became dark red; was blind in both eyes for twenty-four hours, and sight gradually increased to the present amount. Is badly nourished, has dyspnœa. Has stenosis of aortic valves, radial pulse irregular and slow, emphysema of the lungs. Has total loss of color sense in both eyes (no defect formerly). In the right half-fields and in the upper sector of the left half-fields has lost the form sense. In the parts just indicated has light sense, as well as in the rest of the fields. In these parts has subjective flashing which he compares to the rustling of wind in the leaves. Under treatment light sense grew brighter. Had several attacks of headache and vertigo, and temporary blindness. Sight improved until in the eye with corneal opacity vision became $\frac{14}{200}$, in the other $\frac{1}{2}$. He died of heart disease a little more than six months later. The visual fields remained the same.

A case by Brandenberg[3] presented right hemianopia, amnesic alexia of a peculiar kind, viz., he could not read words, but could count figures. The hemianopic loss of sight had respect only to perception of form, but not to

[1] See cases in British Medical Journal, November 23, 1889.
[2] Bericht, 1884, p. 69.
[3] Graefe's Arch. f. Oph., xxxiii., 3, 96, 1887.

perception of light. There was also reduced color sense in the remaining half of the fields. The fourth nerve was paretic and on this account it was believed that a double lesion existed.

The centres for form, light, and color sense are all in the cortex of the occipital lobe. Wilbrand places them one above another; light sense external, form sense intermediate, color sense internal. In this view Reinhardt concurs. Others, viz., Verry, Seguin, and Nothnagel, think them to be side by side. As Swanzy says relative hemianopsia can only occur with lesions of the cortex, hemianopsia from lesions elsewhere must always include all the visual perceptions. Those cases of hemianopsia accompanied with some peripheral contractions of the other side of the field are due, according to Swanzy, to cortical lesions. A case by Oliver[1] indicates that this opinion may be erroneous, and the case is extremely interesting from the precision with which the visual symptoms were studied and made to furnish evidence of localization. The earlier symptoms were headache, vertigo, temporary blindness during two years and then followed by spasm of the hand, arm, and to a less degree of the leg. There was right lateral hemianopsia. Three weeks later the remaining half-fields became extremely contracted, this contraction most decided for the left eye. Vision, $\frac{5}{40}$ O. U. Hemianopic pupillary inaction plainly elicited and showing interference with the sensory motor arc of the pupils. A hemorrhage into the right optic disk. Diagnosis: "Taken in connection with the right hemianæsthesia and right hemiplegia, the ocular changes probably place the lesion near or in the left optic thalamus (pulvinar)." Autopsy revealed a tumor invading the external portion of the left optic thalamus as well as the corpus striatum almost as far as its anterior third. The capsule was not invaded. The left optic tract as far forward as the chiasm was markedly flattened and

[1] Transactions of the American Ophthalmological Society, 1890, p. 479.

pressed upon. The right brain was normal. The tumor was hard, and proved to be a glioma with commencing sarcomatous degeneration. The same remarks may apply measurably to subcortical lesions. But we are apt in such cases to meet with decided distant symptoms in hemiplegia, hemianæsthesia, ocular paralyses, etc.

A symptom often concomitant to occipital lesions on the left side has been referred to, viz., mental blindness and with it amnesic aphasia may concur. It is not necessary to enter into any description of it. But that its locality has been pretty nearly determined is proven by the case of McEwen.[1] A man who had received a severe injury a year previous suffered from deep melancholy and homicidal impulses and paroxysms of headache of indefinite seat. The only localizing symptom was mental blindness, which immediately followed the injury and lasted only two weeks. Acting upon this hint the skull was trephined. It was found that a depressed portion of the inner table of the bone pressed upon the posterior part of the supra-marginal convolution and the anterior corner of the angular gyrus. When the bone was restored to place the mental condition of the man was greatly relieved. This case gives more precision than any yet published to our knowledge of the region in which to look for the visual memory of objects. I do not find any other approaching it in this regard, except one referred to by Gowers,[2] in which a lesion is located at the angular gyrus of the left hemisphere (reported by Chauffard), and is said to have caused complete mind blindness during the short time the patient lived. For a list of cases, nine in number, in which this symptom was present in greater or less degree see Starr, "Familiar Forms of Nervous Diseases," 1890, p. 64. See also "Wilbrand die Seelenblindheit."

A case of Deutschman's [3] has interest. A boy, aged fif-

[1] British Medical Journal, August 11, 1888.
[2] Diseases of the Nervous System, p. 463, American edition.
[3] Beiträge zur Augenheilkunde, 1890, p. 31.

teen, was kicked by a horse upon the left side of the head. Amnesic aphasia occurred, which disappeared in about a week. Then, examined by Professor D——, complete right-sided hemianopsia was found, including light, form, and color sense, and going through the fixation point. In sixteen to eighteen days this passed away and the power of reading became normal. The explanation is thought to be a superficial clot at first compressing the speech centre and settling down afterward upon the visual centre, and when absorbed, function was restored.

Recoveries from hemianopsia are recorded, and even from double hemianopsia. See *British Medical Journal*, Mr. Lang, November 23, 1889. When distant symptoms coexist recovery is not so likely as when these complications are absent.

It would be an unwarrantable omission to say nothing of the behavior of the pupil as aiding the localization of hemianopsia. See Leber in "Graefe and Saemisch Handbook," 1876, Bd. v., iii., 941. Wernicke and Seguin have dwelt upon it. Briefly it is that while in lesions behind the primary ganglia the pupils may act sluggishly, they will respond if light be thrown upon the blind side of the eye. When the lesion is at or in front of the primary ganglia the reaction of the pupil is suspended for the blind halves of the fields.

Swanzy says, "Loss of the pupillary reflex to light, apart from cases of paralysis of the third nerve, is a sign of lesion of the anterior quadrigeminal bodies or of the optic tracts, and may be utilized to distinguish these lesions from others, which may cause loss of sight by implicating both visual paths beyond the corpora quadrigemina, or both visual centres; for in such cases, notwithstanding the amaurosis, the pupil reflex is maintained. Gudden's investigations[1] showed that there are special afferent fibres in the optic nerves and tracts for the pupil-reflex, distinct from those of vision.

[1] Sitzungsbu. d. Münch. Ges. Morphol. u. Physiol., 1886, p. 168.

"In cases of hemianopsia, similarly, the pupil-reflex serves to establish a diagnosis between a lesion in an optic tract and one further on in the visual path, or in the visual centre, of the same side. For, if the pupil contracts actively to light concentrated on the blind side of the field, the lesion cannot be in the tract, but if it does not react, the lesion must be in the tract." [1]

A notable instance which emphasizes this indication is related by Wernicke (the case was seen by Brieger).[2] A girl, aged nineteen, presented the following important symptoms of focal lesions: Slight hemiparesis of left side, anosmia and total blindness, with double choked disks. The pupils moderately dilated and acting imperfectly under diffused daylight. The blindness, therefore, could not proceed from the optic nerves, but from a central cause, and must be regarded as a double hemianopsia. Anosmia could be looked upon, like the slight hemiparesis, as a distant symptom and be left out of view in localization. Tumor of both occipital lobes was the diagnosis. At the autopsy there was found a tumor in the right temporal lobe which had caused softening in the right occipital lobe, but the left occipital was entirely free. This accounted for the left hemianopia. The right hemianopia was explained by finding a blood-vessel across the left tractus, where it had made a deep furrow and given rise to tissue degeneration. A correct diagnosis of the case might have been made during life if the peculiar hemianopic pupillary inaction [3] had been looked for. This can only be made out by examination with a single light in a dark room, and the light must fall quite obliquely lest by diffusion the sensitive side of the retina be stimulated.

I have omitted detailed reference to other symptoms which appear with hemianopsia, such as the various kinds

[1] The Localization of Cerebral Disease, Trans. Oph. Soc. of the United Kingdom, vol. ix., p. 18. 1887.

[2] Tome iii., p. 336.

[3] This phrase is truly descriptive of the facts and to be preferred to "hemiopic pupillary reaction" which is misleading.

of paralysis of sensation and motion. They belong to a discussion of cerebral pathology, and my aim has simply been to give prominence to visual disturbances and point out how a more critical observation may render them more productive of information as to the nature of a cerebral lesion.

The points set forth may be thus summarized :

1. The want of uniformity in the exact location of the boundary between the blind and seeing portions of the fields in hemianopsia.

2. The continuance of a small central field in each eye two or three degrees in diameter, in certain cases of double homonymous hemianopsia.

3. Sectorial defects, homonymous in character, are most likely to have origin in the cortex of the occipital lobe, usually in the neighborhood of the cuneus. In these cases there may be absence of other significant symptoms, except, possibly, agraphia, mental blindness, alexia.

4. Sectorial defects may have origin in the subcortical substance of the occipital lobe, but the defective portion of the fields are not likely to be so well defined in boundaries as in 3, and the loss of light sense will be less complete ; there may even be irrregular spaces where light is recognized, mingled with blind spaces. In these cases hemiplegia, hemianæsthesia, etc., if present, signify proximity of the lesion to the anterior part of the visual radiation of Gratiolet.

5. Sectorial defects, quadrants, or other figures, can arise from lesions of the tractus (cases of Marchand and Norris), but will be accompanied by other significant symptoms pointing to the temporal lobes or to the base, such as paralyses or anæsthesiæ, etc. In these cases, as in all cases of tractus lesion, the blind region includes loss of light-, color-, and form-sense.

6. Loss of either color-sense or of form-sense, with preservation of light-sense, implies lesion of the cortex of the visual centre either, direct or indirect.

7. The hemianopic pupillary inaction signifies that the lesion is at the middle ganglia of the brain, or in front of them, viz., along the tractus, chiasm, or optic nerves.

8. It is certain that a topographical correspondence exists between the cerebral visual centre and the retina, and that it is precise and extends to details. Hun's case proved that the lower part of the cuneus corresponds to the upper part of the opposite halves of the retinæ, and Wilbrand's cases corroborate Hun's, and carry the relationship to smaller regions in the respective localities.

NOTE.—The perimeter of Priestley Smith is, for convenience of manipulation and of registration, to be preferred, while the small hand perimeter of Schweigger is portable and suited to bedside examinations.